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Doppler ultrasound in liver cirrhosis: correlation of hepatic artery and portal vein measurements with model for end-stage liver disease score in Egypt

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Abstract

Background: Liver cirrhosis is a multi-etiological entity that alters the hepatic functions and vascularity by varying grades. Hereby, a cross-sectional study enrolling 100 cirrhotic patients (51 males and 49 females), who were diagnosed clinically and assessed by model for end-stage liver disease (MELD) score, then correlated to the hepatic Doppler parameters and ultrasound (US) findings of hepatic decompensation like ascites and splenomegaly.

Results: By Doppler and US, splenomegaly was evident in 49% of patients, while ascites was present in 44% of them. Increased hepatic artery velocity (HAV) was found in 70% of cases, while 59% showed reduced portal vein velocity (PVV).

There was a statistically significant correlation between HAV and MELD score ($p = 0.000$), but no significant correlation with either hepatic artery resistivity index (HARI) ($p = 0.675$) or PVV ($p = 0.266$).

Moreover, HAV had been correlated to splenomegaly ($p = 0.000$), whereas HARI ($p = 0.137$) and PVV ($p = 0.241$) did not significantly correlate.

Also, ascites had correlated significantly to MELD score and HAV ($p = 0.000$), but neither HARI ($p = 0.607$) nor PVV ($p = 0.143$) was significantly correlated.

Our results showed that HAV > 145 cm/s could confidently predict a high MELD score with 62.50% and 97.62 % sensitivity and specificity.

Conclusion: Doppler parameters of hepatic vessels (specifically HAV) in addition to the US findings of hepatic decompensation proved to be a non-invasive and cost-effective imaging tool for severity assessment in cirrhotic patients (scored by MELD); they could be used as additional prognostic parameters for improving the available treatment options and outcomes.

Keywords: Hepatic, Artery, Portal, Vein, Doppler, MELD, Cirrhosis

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Background

Liver cirrhosis is a worldwide clinical problem that has various infective and metabolic etiologies like hepatitis and alcoholic; the superadded hepatic fibrotic changes and development of regenerative nodules are considered the mainstay culprit of hepatic decompensation and lead to alteration of the hepatic vascular profile that result in portal hypertension [1].

The most commonly utilized (clinical/laboratory) scoring systems for morbidity and mortality prediction in cirrhotic patients are Child-Turcotte-Pugh (CTP) and model for end-stage liver disease (MELD) scores. They could be especially applied for those who would undergo surgeries or interventions. It should be noted that MELD scores of < 10, 10 to 19, and > 19 are relevant to CTP classes A, B, and C, respectively [2–4].

MELD score incorporates three available laboratory tests including the serum creatinine, international normalized ratio (INR), and serum bilirubin, and could be calculated using a certain formula [1, 5].

However, the MELD score had surpassed CTP in its use, being calculated by more objective parameters (laboratory tests) than do the CTP, moreover, the serum creatinine (one of the MELD parameters) had added value in highlighting the importance of the kidney functions in predicting the mortality risk in end-stage hepatic disease patients [2, 3].

Liver biopsy is an invasive procedure that can be employed for the same purpose but still carrying the bleeding risks and is less favorable by a sector of patients. Fibroscan could be used for assessment of the degree of the hepatic stiffness and detection of cirrhosis [6]; however, studying the vascular hepatic parameters by Doppler Ultrasound and the detection of ultrasound findings of hepatic decompensation in cirrhotic patients with correlation to a reference scoring system like MELD could offer an alternative non-invasive investigation that can be done by the available ultrasound machines in the setting of routine abdominal ultrasound scans.

Aim of work

To evaluate the Doppler flow parameters of hepatic vessels and the ultrasound findings of hepatic decompensation and investigate any correlation of observed changes with the severity of liver cirrhosis based on the MELD score system.

Methods

Subjects

A cross-sectional study was carried out in our institute and enrolled 100 cirrhotic patients (51 males and 49 females) who were recruited for primary screening for hepatocellular carcinoma, during the period from

September 2019 to October 2020, with an age range of 25 to 70 years (mean \pm standard deviation [SD], 56.36 ± 7.20).

Inclusion criteria

Patients with liver cirrhosis through a combination of the clinical history and typical sonographic findings, including bright coarse hepatic echotexture, nodular liver surface, and widened interlobar fissures.

Exclusion criteria

The patients who had metastatic disease, hepatocellular carcinoma, or other malignant focal liver masses were excluded from the study.

Written informed consent was obtained from all the patients after explanation of nature and the purpose of our study.

This study was performed after the approval of the local scientific and ethical committee.

All of our patients were subjected to:

1. *Full medical history* with focusing on risk factors especially for viral hepatitis as blood transfusion, dental procedures, also, the history of symptoms of chronic liver disease (CLD) was checked for.
2. *Laboratory investigations*, which included the serum creatinine, international normalized ratio (INR), and serum bilirubin. MELD could be calculated according to the following formula [1, 5]:

$$\begin{aligned} \text{MELD} = & 3.78 \times \ln [\text{serum bilirubin (mg = dL)}] \\ & + 11.2 \times \ln [\text{INR}] + 9.57 \\ & \times \ln [\text{serum creatinine (mg/dL)}] + 6.43 \end{aligned}$$

3. *Doppler and grayscale ultrasound*

- a) Grayscale ultrasound: all patients were instructed to fast for at least 6 hours before the ultrasound scan.

The liver was imaged with a standard grayscale using a 3.5 MHz curvilinear transducer using GE Logiq P7 (GE Healthcare, Waukesha, WI). The splenic length was measured in a longitudinal axis during inspiration in a supine position. The presence or absence of ascites was reported.

- b) Doppler ultrasound was done through the right lateral intercostal or subcostal approaches to detect

and interrogate the hepatic vessels. Peak systolic velocity of the hepatic artery velocity (HAV) was measured in centimeters per second (cm/s) at the hepatic hilum using a corrected Doppler angle of less than or equal to 60° as an angle of insinuation. The Hepatic Arterial Resistive Index (HARI) was automatically calculated by a dedicated software in the machine or could be manually calculated using the following equation if it was needed:

Resistive index (HARI) = [(peak systolic velocity-end-diastolic velocity)/peak systolic velocity] [7].

Portal vein peak velocity (PVV) was measured also in centimeters/second using a corrected Doppler angle of less than or equal to 60°.

4) Statistical methods and data analysis:

Data was entered and processed using SPSS version 24.0 (SPSS IBM). A comparison between two independent mean groups for parametric data was done using the Student's *t* test, while descriptive and inferential statistical analyses were carried out as appropriate [8].

Results are expressed in mean \pm SD, and comparisons with *p* value < 0.05 were considered as statistically significant [9].

Results

The Doppler parameters and ultrasound findings were including the following:

Hepatic artery velocity (HAV)

Our results showed increased HAV in 70% of the study population; thus, it was considered as an indicator of chronic liver disease. (HAV: mean \pm SD, 91.72 \pm 46.26) (Table 1) (Fig. 1a).

Portal vein velocity (PVV)

We had noticed a reduction in the PVV, being depicted in 59.0% of our patients, PVV (mean \pm SD, 19.68 \pm 6.49) (Table 1) (Fig. 1b).

Hepatic artery resistivity index (HARI)

We had reported a lack of significant correlation between the HARI and the severity of cirrhosis and portal

hypertension. HARI (mean \pm SD, 0.64 \pm 0.10) (Table 2) (Fig. 1c).

Spleen size correlation

Out of 100 patients, 49 patients (49%) had splenomegaly by ultrasound examination. Again, there was a significant correlation between HAV and splenomegaly (*P* = 0.000), whereas HARI (ρ = 0.137) and PVV (ρ = 0.241) did not significantly correlate with splenomegaly (Table 3) (Fig. 2a).

Ascites correlation

Ascites was present in 44 (out of 100) of our patients (44%). There was a significant correlation with the MELD score and HAV (ρ = 0.000), but HARI (ρ = 0.607) and PVV (ρ = 0.143) did not show any significant correlation with the presence of ascites (Table 4) (Fig. 2b).

Correlation of Doppler parameters with markers for hepatic decompensation

The MELD score was available in 100 patients (mean \pm SD, 14.09 \pm 5.49; MELD score range 6.00–30.00). In our results, there was a statistically significant correlation between HAV and MELD score (ρ = 0.000), but no statistically significant correlation with either HARI (ρ = 0.675) or PVV (ρ = 0.266). Scatter plots of HAV (Fig. 1a), PVV (Fig. 1b), and HARI (Fig. 1c) as compared with the MELD score were obtained (Table 2).

(HAV: mean \pm SD, 91.72 \pm 46.26); HARI (mean \pm SD, 0.64 \pm 0.10); PVV (mean \pm SD, 19.68 \pm 6.49) (Table 2) (Fig. 1a–c).

Diagnostic performance of elevated HAV in predicting hepatic decompensation

We had suggested that patients with HAV greater than 145 cm/s (the cut-off point) can be confidently predicted to have high MELD scores with a sensitivity of 62.50% and specificity 97.62 % (Table 5)(Fig. 2c, d).

Discussion

Liver cirrhosis is a well-known clinical entity that has a worldwide distribution with no discernable difference in gender and race, albeit having multiple etiologies; it might have the same longstanding consequences and complications like portal hypertension and hepatic decompensation. It had been agreed that the gold standard reference for the assessment of the degree of cirrhosis is the histological analysis obtained by liver biopsies; however, it should not be the standard care for all cirrhotic patients, being an invasive procedure especially in patients with hemorrhagic tendency [10].

As liver cirrhosis continues, it may alter the hepatic vascular parameters; thus, the Doppler ultrasound scan

Table 1 The percentage of hepatic artery and the portal vein velocity thresholds in our patient population

		No.	%
Hepatic artery velocity	Decreased (< 30 cm/s)	2	2.0%
	Normal (30–60 cm/s)	28	28.0%
	Increased (> 60 cm/s)	70	70.0%
Peak velocity of portal vein	Decreased (< 20 cm/s)	59	59%
	Normal (20–40 cm/s)	34	34%
	Increased (> 40 cm/s)	7	7%

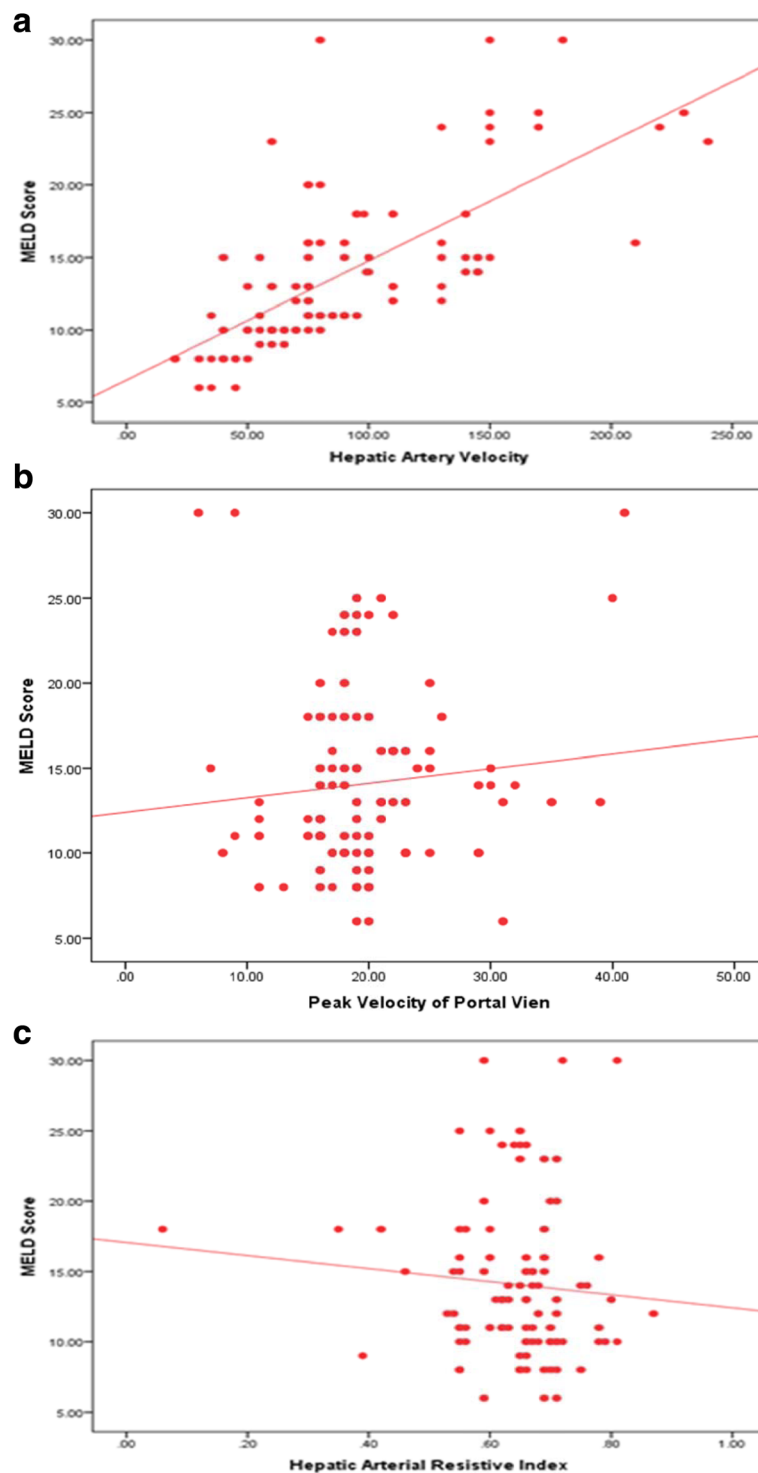


Fig.1 **a** Scatter plot of MELD score as a function of HAV (cm/s), the solid line represents the best-fit linear regression. **b** Scatter plot of MELD score (y-axis) as a function of PVW (cm/s) (x-axis). The solid line represents the best-fit linear regression. **c** Scatter plot of MELD score (y-axis) as a function of HARI (x-axis). The solid line represents the best-fit linear regression

is warranted as a primary focus for many researchers to be an alternative investigation that could surrogate such invasive procedures [10].

As we mentioned, MELD score system had outperformed the CTP system in patients with chronic liver disease especially those who are candidates for hepatic

Table 2 Correlation between Doppler ultrasonography findings and MELD score

		MELD < 19 No.=84	MELD > 19 No.=16	Test value•	P-value	Sig.
Hepatic Artery Velocity	Median (IQR)	75 (55 – 100)	150 (80 – 175)	-4.092	0.000	HS
	Range	20 – 210	60 – 240			
Hepatic Arterial Resistive Index	Median (IQR)	0.66 (0.6 – 0.7)	0.65 (0.61 – 0.71)	-0.419	0.675	NS
	Range	0.06 – 0.87	0.55 – 0.81			
Peak Velocity of Portal Vein	Median (IQR)	19 (16 – 22)	19 (18 – 23.5)	-1.113	0.266	NS
	Range	7 – 39	6 – 41			

$P > 0.05$ non-significant (NS); $P < 0.05$ significant (S); $P < 0.01$ highly significant (HS) • independent *t* test, • Mann-Whitney test

transplantation, being more based on objective laboratory biomarkers than do the CTP system; moreover, MELD score does not consider demographic factors like age and race in hepatic transplantation listing; thus, it had been considered as a more favorable scoring system than CTP [1, 3, 10].

On review of literature, the main objective of this study was to evaluate whether there was a correlation between hepatic Doppler parameters as well as the ultrasound findings of hepatic decompensation with the clinical status in cirrhotic patients as measured by MELD score.

In light of our results, we found a statistically significant correlation between the hepatic artery velocity (HAV) and MELD score (Tables 1 and 2) (Figs. 1a, 3, and 4), where 70% of our cases had showed increased HAV.

This was concordant with Park et al. studies who involved 264 cirrhotic patients and had correlated the Doppler parameters with markers for hepatic decompensation and reported a statistically significant correlation between HAV and MELD score ($\rho = 0.0001$) [11, 12]; Glisic et al. had also concordant supportive results, but with a relatively smaller sample size (80 patients and 20 controls), and they had reported an increase in HAV of cirrhotic patients with an average (125.7 cm/s),

compared to their healthy controls (79.3 cm/s) [13]; our results had matched theirs, where the cut-off point was 145 cm/s (Table 5 and Fig. 2c, d).

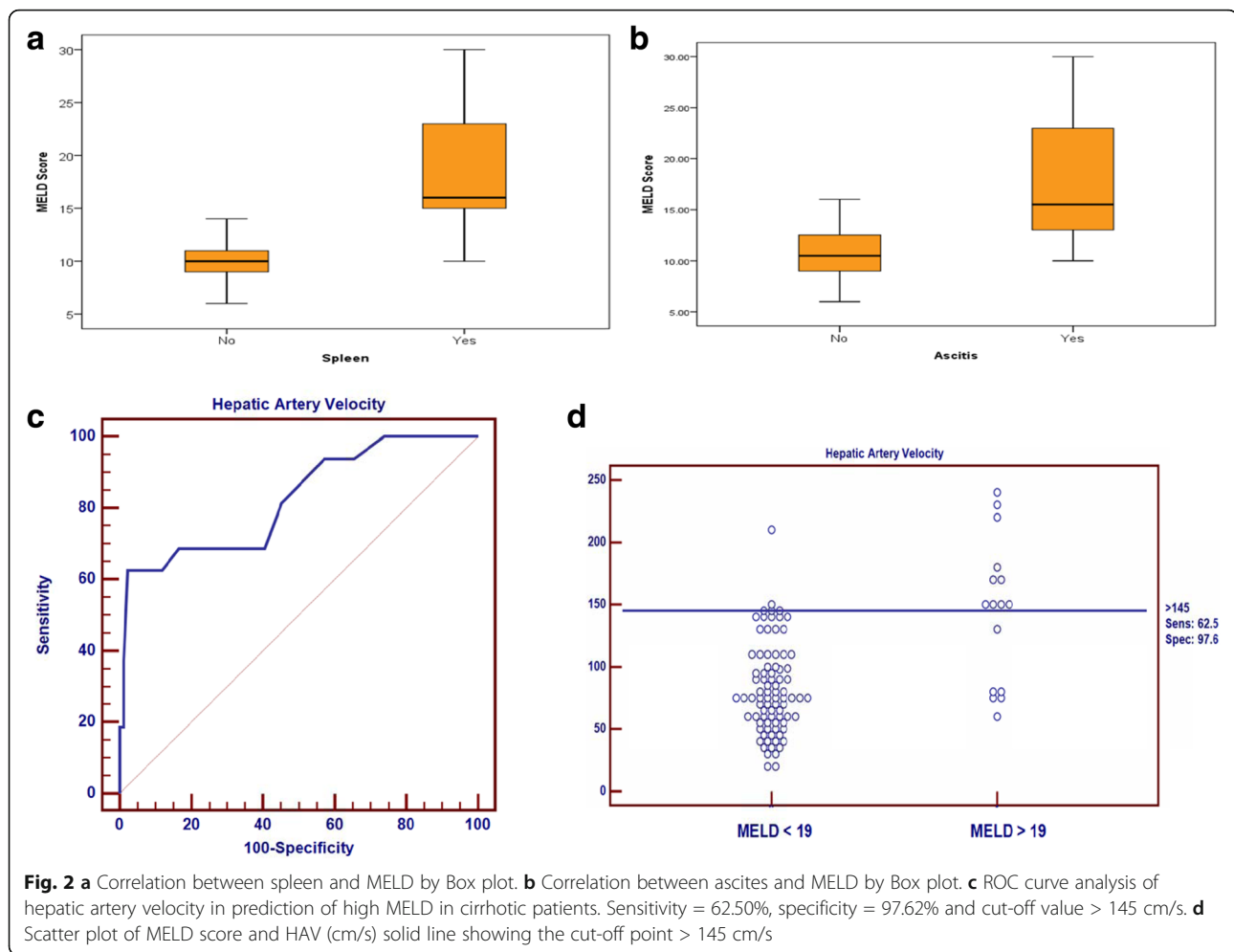
A literature-based rationale for such results, where the increased hepatic sinusoidal resistance would, in turn, decrease the portal venous inflow; therefore, it activates the hepatic artery buffer response (HABR; mediated by adenosine washout and angiotensin II system) and increases the hepatic arterial inflow as a compensatory mechanism, to maintain consistent oxygen in the hepatic parenchyma; nevertheless, some studies had documented that HABR is maintained in the setting of cirrhosis [14, 15].

Discordance with Azizah et al. study was present in this regard, where they included 56 eligible cirrhotic patients and studied the correlation of hepatic hemodynamics with CTP score, and found that the HAV was not significantly different between the study and the control groups ($\rho = 0.06$). Their possible reason for the contradicting data was that two different US systems were used to scan both control and study groups. No prior checking was carried out by scanning the same patient/s using these two US scanners to look for any significant difference in the measurement of the studied parameters. Also, they were using the CTP score in classifying their patient population, assessing their

Table 3 The spleen size correlation with MELD and the hepatic Doppler parameters

		Spleen		Test value•	P-value	Sig.
		No	Yes			
		No. = 51	No. =49			
MELD Score	Median (IQR)	10 (9 – 11)	16 (15 – 23)	-8.054	0.000	HS
	Range	6 – 15	10 – 30			
Hepatic Artery Velocity (HAV)	Median (IQR)	65 (50 – 80)	110 (80 – 145)	-5.754	0.000	HS
	Range	20 – 145	35 – 240			
Hepatic Arterial Resistive Index (HARI)	Median (IQR)	0.66 (0.61 – 0.71)	0.65 (0.59 – 0.69)	-1.485	0.137	NS
	Range	0.39 – 0.87	0.06 – 0.81			
Peak Velocity of Portal Vein (PVV)	Median (IQR)	18 (16 – 20)	19 (17 – 23)	-2.048	0.241	NS
	Range	7 – 39	6 – 41			

$P > 0.05$, non-significant (NS), $P < 0.05$ significant (S), $P < 0.01$ highly significant (HS), • independent *t* test, • Mann-Whitney test



prognosis, and to predict their mortality. An important deficiency in the CTP score is that it uses 2 very subjective variables in its calculation, namely portosystemic encephalopathy and severity of ascites, again, MELD score calculation is based on more objective parameters [16].

Portal flow velocity was also a studied parameter, although 59% of our patients showed significantly decreased portal flow velocity (< 20 cm/s) (mean \pm SD, 19.68 ± 6.49) (Tables 1 and 2) (Figs. 1b, 3, and 4). We had found that PVV did not significantly correlate with

Table 4 The presence of ascites and correlation with MELD and other Hepatic Doppler parameters

		Ascites		Test value•	P-Value	Sig.
		No No. = 56	Yes No. = 44			
MELD Score	Median (IQR)	10.5 (9 – 12.5)	15.5 (13 – 23)	-6.239	0.000	HS
	Range	6 – 25	10 – 30			
Hepatic Artery Velocity	Median (IQR)	70 (50 – 90)	100 (75 – 140)	-3.920	0.000	HS
	Range	20 – 210	40 – 240			
Hepatic Arterial Resistive Index	Median (IQR)	0.66 (0.6 – 0.7)	0.65 (0.6 – 0.7)	-0.515	0.607	NS
	Range	0.35 – 0.87	0.06 – 0.81			
Peak Velocity of Portal Vein	Median (IQR)	19 (16.0 – 20.5)	19 (17.5 – 25.0)	-1.829	0.067	NS
	Range	7 – 31	6 – 41			

P > 0.05 non-significant (NS), P < 0.05 significant (S), P < 0.01 highly significant (HS), • independent t test, • Mann-Whitney test

Table 5 The cut-off point, sensitivity and specificity of HAV thresholds to MELD

Cut-off point	Sensitivity	Specificity	+PV	−PV
> 145 cm/s	62.50	97.62	83.3	93.2

MELD score; this was concordant with many works of literature [16–19] who had documented the same issue; however, this was contradicting the Berzigotti et al. study, where they reported a low flow velocity of < 20 cm/s in addition to a caliber increase in the main portal vein (MPV) were considered diagnostic features of portal hypertension [20]. However, we did not measure the portal vein diameter, and the possible reason for the contradicting data in the PVV was the heterogeneity of the cirrhotic patients included in various studies, also the type of hemodynamic disturbances, for example, the presence of hyperkinetic syndrome or portosystemic collaterals.

Hepatic artery resistivity index (HARI) was additionally investigated in our present study. We found that HARI did not significantly correlate with MELD score, splenomegaly, or ascites (Tables 2, 3, and 4) (Fig. 1c).

This was matching the study done by Iranpour et al., who had observed a specificity and sensitivity of utilizing a hepatic arterial resistive index (RI) cut-off of > 0.77 to predict cirrhosis were as low as 70% and 68%,

respectively [15], further supportive results had been provided by other studies [7, 11, 13].

This was discordant with the Azizah et al. study, where they found that the increased HARI in the liver cirrhosis was statistically significant and it helped in differentiating cirrhotic participants (where HARI values were above 0.7) from the non-cirrhotic ones (HARI values were less than or equal to 0.7). A possible explanation for the contradicting data is that the RI of the hepatic artery can be changed by certain physiological factors and could be elevated shortly after meal ingestion, where the portal vein flow increases then a corresponding reduction in the arterial flow was induced by vasoconstriction. Nevertheless, the state of vasoconstriction could result in a transient elevation of RI that could last up to few hours [21, 22]; this is why the HARI should be carefully obtained with special consideration for the physiological variations. In our study, we did instruct all patients for 6 h fasting to avoid this diagnostic bias and we had found no significant correlation.

In the setting of cirrhosis, findings like splenomegaly and ascites could suggest a state of hepatic decompensation, we had depicted a statistically significant correlation between the presence of splenomegaly, MELD score, and HAV; however, this significant correlation was not existing with either PVV or HARI (Table 3)(Figs. 2a, 3, 4). At this extent, we had a concordance

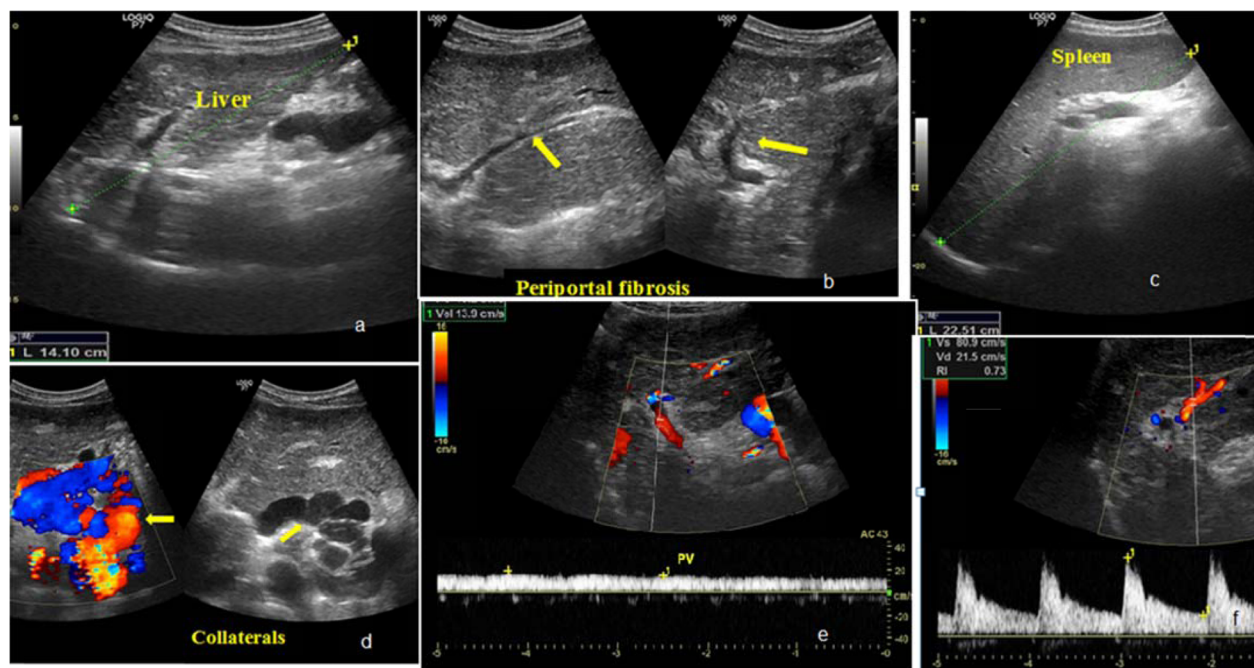
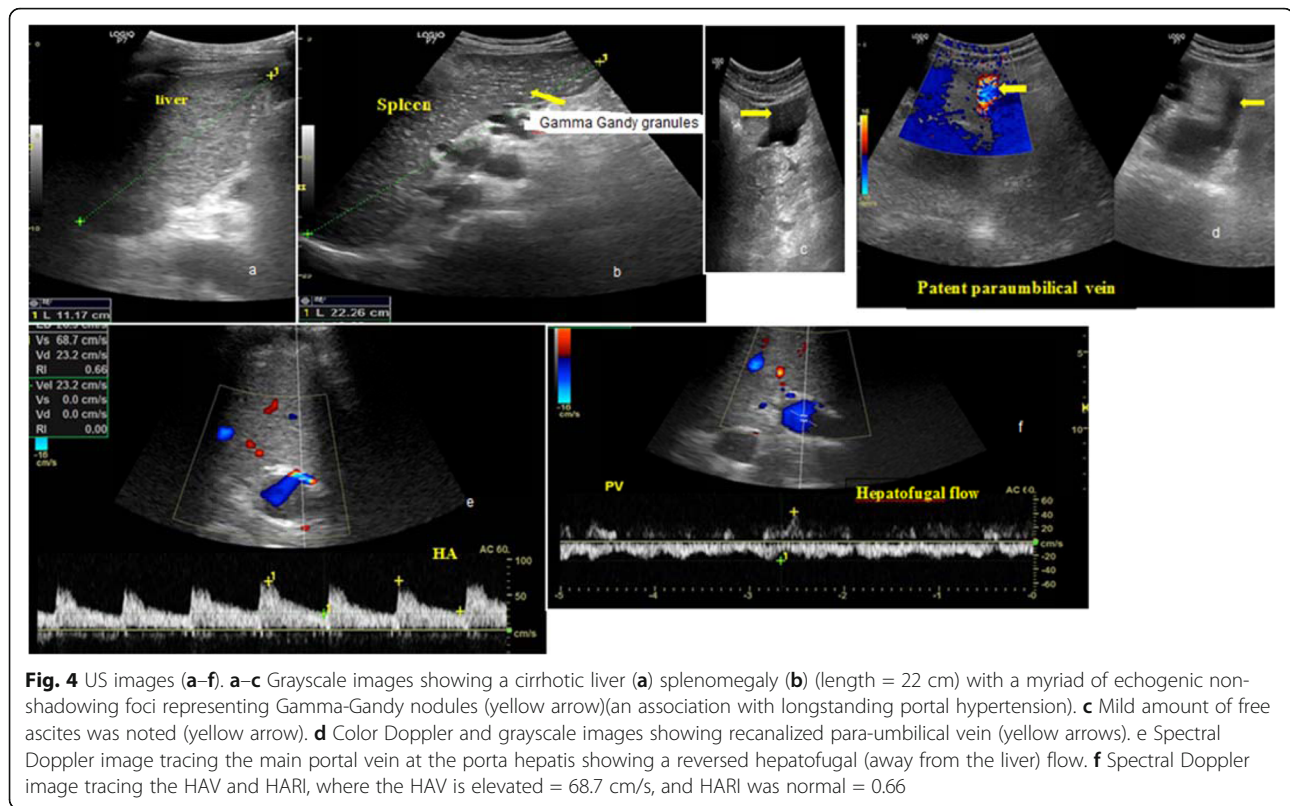


Fig. 3 US images (a–f). **a, b** Grayscale images showing a cirrhotic liver with uneven surface and periportal fibrosis (yellow arrows). **c** Grayscale image showing splenomegaly (length = 22 cm). **d** Color Doppler image showing a cluster of gastro-esophageal porto systemic collaterals (yellow arrows). **e** Spectral Doppler image tracing the PVV that was 13.9 cm/s (reduced) [Normal = 20–40 cm/s]. **f** Spectral Doppler image tracing the HAV and HARI, where the HAV is elevated = 80.9 cm/s [Normal = 30–60 cm/s], it also shows abnormal high HARI = 0.73 [Normal = 0.60–0.68]



with many studies [11, 23–25], but on the other hand, it was discordant with the Yadav et al. study [26], who found (out of 112 patients) no significant correlation between MELD score and spleen size in cirrhosis.

Kashani et al. had explained this discordance, where the size of the spleen could vary between cirrhotic patients according to their primary disease etiologies, with hepatitis C virus (HCV) infected and non-alcoholic hepatitis patients were showing significantly larger organ dimensions as compared to those with alcoholic hepatitis [27], almost all of our patients lie in the category of post-infection (hepatitis) cirrhosis, where the splenomegaly was accordingly a harmonious feature. It worth mentioning, that some authors had suggested that splenomegaly is associated with a poor prognosis in cirrhotic patients and could be used as an index for the non-invasive assessment of esophageal varices and bleeding risks during radioactive or acoustic examinations [28, 29].

Ascites is another important complication of cirrhosis and is usually associated with poor prognosis (1- and 5 year mortalities are 15% and 44%, respectively), on follow-up [30].

The current study showed a statistically significant correlation between the presence of ascites and the MELD score as well as HAV. But on the other side, the correlation with both PVV and HARI (Tables 2 and 4)

(Figs. 2b and 4) was statistically insignificant; this was in line with Azizah et al., Park et al., and Somsouk et al. studies [11, 16, 30].

Yet, Popov et al. had evaluated and compared the changes of hepatic and renal Doppler parameters in cirrhotic patients, and found no significant differences between hepatic Doppler parameters and the presence or absence of ascites [31]. Our explanation for this mismatch was attributed to the different stages of hepatic cirrhosis in their patients who could have a previous tapping or had received treatments for ascites. But most of our ascetic patients had a decompensated end-stage liver disease and gave no history of previous surgical intervention or the transjugular intrahepatic portosystemic shunt (TIPS) to treat their ascites.

Although providing potentially useful data, we had some limitations in this work including the following:

First, in this study, we did not correlate the degree of cirrhosis or confirm the presence of cirrhosis on histologic bases. Although we agree that histology is considered a reference standard, it was not the standard protocol to have a hepatic biopsy in all cirrhotic patients. Second, we did not include normal controls in our study, as we consider that the MELD score has only been used in cirrhotic patients and is not validated to be used in normal individuals. Moreover, normal individuals do not have regularly checked laboratory tests for MELD to

provide a fair comparison. Third, is that our study sample was from a single center. Finally, the study duration was relatively short (about 12 months) with a relatively small sample size of recruited patients; therefore, statistical analysis may not be so robust.

Conclusion

Doppler US of hepatic vessels (specifically the HAV) in addition to the ultrasound findings of hepatic decompensation like splenomegaly and ascites had proved to be a non-invasive and cost-effective imaging tool for assessment of the severity in chronic liver disease patients (as scored by MELD); they could be used as additional prognostic parameters for improving the available treatment options and outcomes.

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Not applicable

Authors' contributions

AAB: the corresponding author had contributed by supervising the ultrasound examinations and in the final editing and submission of the manuscript. RMM had done the ultrasound examinations for the patients and shared in the manuscript editing and reference collection. KHE: had introduced the idea of the current study and helped in the image selection and revised the final version of the submitted manuscript. All author(s) read and approved the final manuscript.

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Availability of data and materials

All data are available on a software system owned by each of the authors and the corresponding author has the authority to respond if there is any query.

Ethics approval and consent to participate

The protocol was reviewed and approved by the local ethics committee of the radiology department, Kasr Alainy Hospital, Cairo University. The reference number: Code MS-63_2019, date of approval: 28 September 2019. All patients had given their written consents to participate in this work.

Consent for publication

All patients had given their written consents for publication of this work.

Competing interests

All authors declare that they have no competing interests.

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